



On the pathogenesis of anterior polar cataract.

By E. TREACHER COLLINS.

(With Plate I.)

CATARACT at the anterior pole of the lens results from a degeneration and breaking up of lens fibres in that position, together with proliferation of the overlying intra-capsular epithelium.* It most commonly forms in cases of perforating ulcer of the cornea, but may in infants' eyes follow ulceration of the cornea without perforation.

Two views have been put forward as to the way in which ulceration of the cornea excites changes at the anterior pole of the lens. Hulke suggested that it was due to contact of the lens and cornea arresting the osmose of nutritional fluid through the anterior capsule. He pointed out that in an infant's eye the anterior chamber is so exceedingly shallow that the swollen condition of an inflamed cornea might be quite sufficient to bring them into contact apart from any perforation.

Hutchinson's view was that the close proximity of the inflamed area in the cornea to the anterior pole of the lens could, by "a kind of vital catalysis," excite changes in it. He objected to Hulke's theory on the ground that there was a proliferation of cells which he thought inconsistent with an arrest of nutrition.

* *Vide* paper on the "Minute Anatomy of Pyramidal Cataract," 'Trans. Ophth. Soc.,' vol. xii, p. 89.

To meet this objection I have demonstrated* that besides the proliferation of capsule cells there is some degeneration of lens fibres, and that the probable course of events is—1st, arrest of osmose of nutritional fluid by contact of lens and cornea; 2nd, shrinking and degeneration of lens fibres with some softening of lens capsule, and consequent decrease of the tension within the capsule in the affected locality; 3rd, proliferation of the capsule cells where the tension within the capsule is decreased.

It has since occurred to me, that evidence in favour of one or other of these theories might be obtained by finding out if contact of a sarcomatous growth with the lens capsule, in the part lined by cells, excited similar changes.

For this purpose I looked through the sections of a series of cases of sarcoma of the iris and ciliary body. In most cases of sarcoma of the ciliary body where the growth touches the lens it does so at the posterior and lateral part. I have, however, met with one specimen of melanotic sarcoma involving the anterior part of the ciliary body and iris, in which the tumour pressed on the antero-lateral part of the lens. Where the two are in contact the superficial lens fibres have undergone degeneration, and overlying the degenerated area the intra-capsular epithelial cells have proliferated, forming a patch of laminated tissue similar to that met with in anterior polar cataracts (Plate I, fig. 1). That contact of an intra-ocular growth with the lens capsule may excite proliferation of the intra-capsular epithelium had not escaped the notice of that acute observer, Otto Becker, who refers to it in his monumental work on the lens.

This fact, then, observed by Otto Becker, of which I have been able to bring before you a typical example this evening, favours strongly the theory that anterior polar cataract is due to an arrest in osmose of nutritional fluid through the capsule, for in the case of contact with a

* 'Researches into the Anatomy and Pathology of the Eye,' 1896, p. 14.

PLATE I.

Mr. E. Treacher Collins' paper on the Pathogenesis of Anterior Polar Cataract.

FIG. 1.—Melanotic sarcoma of the ciliary body, pressing on the side of the lens, and causing there a subcapsular patch (o) similar in appearance to an anterior polar cataract.

FIG. 2.—Section of a congenitally malformed lens. It is much flatter from before backwards, and has a dense anterior polar opacity.

FIG. 3.—Section of a lens with an anterior polar cataract of eleven years' formation. Patch of degenerate lens substance (o) deeper than mass at anterior pole, which probably gave rise to a second opacity.



Fig. 1.



Fig. 2.



Fig. 3.



sarcoma, it is obvious, there can be no question of vital catalysis from some neighbouring inflammatory process.

In early foetal life, before the secretion of the aqueous humour has commenced, the lens receives nutriment from a network of blood-vessels which surround it, forming the fibro-vascular sheath; the anterior part of this sheath lies between the lens and the back of the cornea. On the opening out of the anterior chamber by the secretion of the aqueous humour the lens becomes separated from the back of the cornea, and the fibro-vascular sheath disappears.

Should the opening up of the anterior chamber for some reason be delayed until after the disappearance of the fibro-vascular sheath, then in the region where the lens and cornea remained in contact the nutrition of the former would be affected, and changes would be expected to occur similar to those produced by contact of lens and cornea from ulceration.

I have already demonstrated to this Society * a congenital anterior polar cataract occurring in association with congenital aniridia, which presented precisely the same microscopical appearances as the anterior polar cataracts produced after birth by ulceration of the cornea.

I have to-night to show you the drawing of the microscopical appearances of another congenital anterior polar cataract, which occurred in an eye having congenital buphthalmos and anterior synechia of the iris (Plate I, fig. 2).

It is a matter of common clinical experience that in performing the operation of discission in some cases of congenital cataract the lens is found not to present its normal dimensions, but to appear shrunken or flattened antero-posteriorly. In the centre of some of these apparently flattened cataracts a more or less raised, dense white, central nodule is observed. This drawing represents the section of a cataract which I believe, had it been possible to examine it during life, would have presented such an appearance. In the centre, immediately behind the much-wrinkled

* 'Trans. Ophth. Soc.,' vol. xiii, p. 128.

anterior capsule, is a large laminated mass, such as is produced by proliferation of the capsule cells in anterior polar cataracts. Beneath this large, conical, laminated mass are signs of extensive degeneration of the lens fibres. So extensive has this degeneration been that the lens is much flattened from before backwards, and the anterior polar laminated mass extends backwards in the centre as far as the posterior capsule.

In a former communication to this Society I mentioned that cases of anterior polar cataract are occasionally met with in which, at a little depth in the lens substance, a second opacity is seen.

In some lectures I gave at the College of Surgeons I was able to quote three cases illustrative of this condition, in all of which several years had elapsed between the inflammatory attack, which was presumably the initial cause of the anterior polar opacity, and the time the patient came under observation.

I have suggested that the explanation of the two opacities is to be found in the increase in the size of the lens, the new formation of cortical lens fibres which occurs separating the mass at the anterior pole, formed by the proliferated cells, from the degenerated lens substance underlying it.

Since then I have met with four more cases presenting a similar appearance ; of these I append brief notes.

CASE 1.—Sophia B—, æt. 48, attended at the Moorfields Hospital on April 30th, 1896. She stated that her mother had told her that she had had inflammation of her eyes when a baby, and that there had been specks on them ever since. In the right eye there was a white opacity at the anterior pole of the lens, and behind it, a little depth in the lens, a second opacity, separated from the first one by some clear lens substance.

In the left eye there were two similar opacities in the lens, but they were not completely separated by clear lens substance, a thin opaque band still uniting them.

Both corneæ were clear.

$$V. \begin{cases} R. \frac{6}{60} \text{ Hm. } 4 \frac{6}{12} \text{ partly} + 5 \text{ J. } 1. \\ L. \frac{6}{60} \text{ Hm. } 4 \frac{6}{12} \text{ partly} + 5 \text{ J. } 1. \end{cases}$$

CASE 2.—William B—, æt. 10, came to the Belgrave Children's Hospital on August 24th, 1897. His mother stated that his eyes were inflamed in infancy, and discharged a great deal.

A faint nebula was seen in the centre of each cornea, and there was a small white dot-like opacity at the anterior pole of each lens. In the right eye there was a second opacity in the lens, in a line with the one at the anterior pole, and separated from it by some clear lens substance.

In the left eye there was a faint mark, in the same position as the deeper opacity in the right lens, hardly itself sufficient to be described as an opacity. V. in each eye = $\frac{6}{36}$ unimproved.

CASE 3.—Henry Richard D—, æt. 60, came to the Moorfields Hospital on December 19th, 1897. He stated that his eyes were inflamed in infancy, and that his sight had always been defective. There were nebulæ of both corneæ.

In the right lens there was a dense white opacity at the anterior pole, which was bifurcated at its margins.

In the left lens there were two dense white circular opacities at the anterior pole, one deeper than the other; they seemed to be united in the centre. There was also a faint grey haze deeper than the deepest white patch. V. in each eye: $\frac{6}{0} \bar{c} - 15 \frac{6}{60}$.

CASE 4.—Richard L—, æt. 44, came to the Moorfields Hospital on December 13th, 1897. He stated that his eyes were inflamed when he was eighteen months old, that his sight had always been defective, and that the left eye was operated on in 1880. There were nebulæ in both corneæ. In the right eye there was a dense white opacity at the anterior pole of the lens, and behind it,

separated by some clear lens substance, was a second faint grey opacity. In the left eye there was a coloboma of the iris down and in; a dense white opacity at the anterior pole of the lens, but no second deeper opacity. V. of R. = $\frac{6}{60}$, of L. = counting fingers.

In all these four cases, as in those previously referred to, an interval of several years had elapsed between the inflammatory attack and the time the patient came under observation.

In the left eye of Case 1 and in both eyes of Case 3, the separation of the deeper from the superficial opacity was incomplete. In the left eye of Case 1 a thin band remained connecting them. In the right eye of Case 3 it cannot be said that there are two opacities, the edge only showing bifurcation. Seeing that the formation of a second opacity in the lens in connection with anterior polar cataract is not very uncommon, it occurred to me that if I looked through my collection of sections of lenses with anterior polar cataracts, possibly I might meet with one showing changes situated a little depth in the lens likely to give rise to a second opacity. This I have succeeded in doing. The case is one of which the details have already been published in the 'Transactions' of this Society, vol. xii, pp. 99—101. In it, eleven years had elapsed between the formation of the anterior polar opacity and the time at which the eye was removed. At the anterior pole of the lens immediately beneath the hyaline capsule is a mass of nearly homogeneous but faintly laminated material, which has patches of epithelial cells embedded in it (Plate I, fig. 3). This mass is slightly raised above the level of the rest of the lens, and the lens substance is slightly cupped beneath it. A thin complete layer of hyaline capsule, lined by epithelial cells continuous with those lining the capsule elsewhere, separates the mass at the anterior pole from the lens substance beneath it. The lens fibres immediately underlying the anterior polar mass appear normal, but a little depth in there is an area over which they have become broken up into irregular

amorphous granules and detritus. This area corresponds fairly accurately in shape to that of the mass at the anterior pole, and I think there can be no doubt that they were at one time in contact, having become separated by the gradual growth of new lens fibres inwards between them.

(*June 9th, 1898.*)







